

REMARKS

Claims 12 - 21, 31, 36, 41 and 50-63, 65, 67-69, 71-73 and 75 -104 are pending. Claims 18-21, 31, 41, 68, 69, 71-73 and 75-97 are allowed. Objection is made to Claims 16, 65, 67, 101 and 104. Claims 12-15, 17, 50-63, 98-100, 102 and 103 are rejected and the rejection is made final.

This response is submitted as the second submission under 37 C.F.R. §1.129. Accordingly, the final rejection should be withdrawn and prosecution resumed in accord with §1.129.

In the above amendment, claim 16 is cancelled in favor of claim 18, which has been allowed. New claims 105 - 108 are added to restore the subject matter of original claims 12-15, which had been allowed previously, and which were designated as not corresponding to the lost interference count.

Claims 12-15 are rejected under 35 U.S.C. 102(g) or, in the alternative, under 35 U.S.C. 103(a) as being drawn to the same invention as the lost count or on the grounds of estoppel. Applicants respectfully disagree.

The interference count [count 2 which was substituted for count 1] is set forth in the Order Redeclearing Interference, Paper No. 108 mailed on May 18, 2001, as follows:

A composition according to claim 1 of Inglis '261 or any of claims 1 or 24 of Inglis '362 or ~~any of claims 1, 5, 9, 25, 42-45~~ **claim 49** of Knipe

or

a method according to any of claims 20, 24 or 41 of Inglis '261 or claim 13 of Inglis '361 [*sic*] or any of claims ~~12, 17, 18, 32 or 37~~ **35 or 40** of Knipe.

Knipe claims which correspond to Count 2 were set forth as claims 1-9, 25-27, 29, 32-35, 37-40 and 42 -49.

Further, Knipe claims not corresponding to Count 2 were set forth on page 49 as claims 12-22, 31, 36 and 41.

However, on page 49 of the Memorandum Opinion and Order, Paper No. 107 also mailed May 18, 2001, the Board further stated: "In effect, Count 2 provides Knipe the relief sought insofar as it omits Knipe claims 1, 5, 12, 17 and 18 from Count 2."

Knipe claim 1 is directed to a "herpesvirus having a mutation in one or more genes encoding a protein essential for viral replication to render the herpesvirus replication defective, said mutant having an ability to effect an antibody subclass shift of IgG2a/IgG upon *in vivo* administration to a mammal."

Knipe claim 5 is directed to a "herpesvirus having a mutation in one or more genes encoding a protein essential for viral replication to render the herpesvirus replication defective, said mutant having an ability to induce production of IFN- γ upon administration to a mammal."

All of the Inglis claims defining the interference Count 2 appear to be directed to a composition or method wherein a

mutant virus is able to cause production of infectious new virus particles in a recombinant complementing host cell expressing a gene which complements said essential viral gene, but is unable to cause production of infectious new virus particles when said mutant virus infects a host cell other than said recombinant complementing host cell.

As testified by Dr. Stephen C. Inglis during the interference [set forth at p. 36 of Memorandum and Order], "Knipes mutants do not produce new progeny virus particles when they infect

normal host cells because they do not produce the structural components (e.g., viral DNA and/or structural proteins, such as capsids) needed to make progeny virus particles."

Further, the Board stated (p. 49) "as to Knipe claims 12-22, methods of using a composition based upon its unknown specific property, i.e., the ability to effect an antibody subclass shift of IgG2a/IgG (claims 12-17) and/or to induce production of IFN- γ upon in vivo administration to a mammal, would not have been obvious unless that composition were already known in the prior art to be useful for that same purpose."

Current claims 12 - 15 are directed to a method wherein "said mutant herpesvirus [has] an ability to effect an antibody subclass shift of IgG2a/IgG upon *in vivo* administration to said mammal." This is a previously unknown specific property. Thus, as held by the Board during interference, the ability to effect an antibody subclass shift of IgG2a/IgG would not have been obvious.

The lost interference Count 2 *fails* to teach that a mutant herpesvirus has an ability to effect an antibody subclass shift of IgG2a/IgG upon *in vivo* administration to a mammal. thus, claims 12-15 are patentable over the lost interference count.

Claims 12-15 and 17 are rejected under 35 U.S.C. §112, first paragraph. The examiner states that the specification does not reasonably enable a method of treating an unspecified condition. Applicants strongly disagree.

Claims 12-15 are directed to a method for **eliciting** an immune response in a mammal. they are not directed to **treating** an unspecified condition. The specification certainly enables the method for **eliciting** an immune response in a mammal.

Regarding Claim 17, it is directed now to a method for treating herpetic encephalitis.

Thus, present claims 12-15 and 17 are fully enabled.

Claims 50 - 63, 98-100, 102 and 103 are rejected under 35 U.S.C. §102(g) or, in the alternative, under 35 U.S.C. §103(a) over the lost count or on the grounds of estoppel. It is believed that the above amendment results in claims that are patentable over the lost count.

Claim 36 is rejected under 35 U.S.C. §102(b) over Dobson et al (Neuron 5:353-60, 1990). Dobson describes a genetically engineered herpes simplex virus having a deletion in the early intermediate gene ICP4, which inhibits replication. There is no teaching or suggestion for a "herpesvirus comprising a mutation in one or more early genes encoding a protein essential for viral genome replication to render the herpesvirus replication defective." It is respectfully submitted that "a deletion in the early intermediate gene ICP4, which inhibits replication" is not a teaching or suggestion for "a mutation in one or more early genes encoding a protein essential for viral genome replication to render the herpesvirus replication defective."

The examiner considers the term early to include ICP4. However, Dobson clearly indicates a difference between the ICP4 gene and early genes by stating (p. 353, bottom of first column through top of second column) that

the immediate early ICP4 gene encodes a protein that activates the transcription of most of the other HSV-1 genes. Deletion mutants in this gene express only four immediate early genes; they do not express early or late RNA . . . (Emphasis added.)

Thus, even Dobson does not consider IPC4 to be an early gene.

Consequently, it is not seen how the present invention is anticipated by, or would have been obvious to one of ordinary skill in the art in view of, Dobson.

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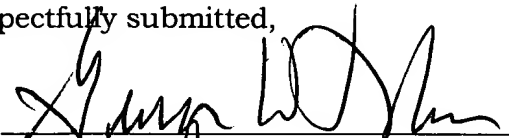
It is respectfully submitted that the subject application is in a condition for allowance. Early and favorable action is requested. If any issues remain, the Examiner is requested to call Applicants' undersigned attorney to expedite the resolution of such issues.

If for any reason a fee is required, a fee paid is inadequate or credit is owed for any excess fee paid, the Commissioner is hereby authorized and requested to charge Deposit Account No. **04-1105**.

Respectfully submitted,

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By:


George W. Neuner
Registration No. 26,964

EDWARDS & ANGELL, LLP
P.O. Box 55874
Boston, MA 02205
(617) 439-4444

Customer No. 21874